b.—PATHOLOGY OF THE NERVOUS SYSTEM AND MIND, AND PATHOLOGICAL ANATOMY.

TETANUS.—The following is a notice of a recent *brochure* by G. Richelot, taken from *La France Médicale.*

In an introduction M. Richelot defines traumatic shock as a violent action exercised at the periphery, then radiating to the centres and reflected, or not, again to the periphery. According to him, this term traumatic shock comprehends all the nervous accidents from wounds; the primary and secondary pain, constituting a traumatic neuralgia, accompanied or not with reflex phenomena, secondary spasms, and true tetanus. This secondary irradiation from the centre to the periphery, may show itself in complex pheaomena, such as the delirium of alcoholism, or from wounds, or the stupor which follows severe crushing of the limbs. According to the author, therefore, tetanus fills a very well marked place in this class of affections. He ranks it among the secondary spasms from traumatic shock, and he makes it a pathological reflex act, involving all the voluntary muscular system.

To determine the true nature of tetanus, the author investigates its etiology, pathological anatomy and physiology. In the etiology he investigates the wounds, the person injured, and the medicine. The pathological anatomy reveals one general fact, congestion of the nerve centres, the nerves, the pharynx, the stomach, and the lungs, due to the muscular efforts, or to asphyxia.

As regards the pathological physiology, two theories include all the ideas emitted in regard to the nature of these convulsions, the muscular theory and nervous theory. The author briefly sums up the first of these as follows: tetanus is a disease of the muscular tissue, it is a contracture of peripheral origin, the primordial lesion of which is in the fibro-conjunctive tissue that surrounds the muscular fibre, and the general morbid element is catarrho-rheumatismal in its nature. The nervous theory may be subdivided into two subdivisions, the purely nervous theory and the humoral theory. The first admits nervous irradiation, but rejects any infection of the blood. The second is also nervous and not at all in opposition to the reflex theory; the cord, according to it, is placed in such conditions by the toxic agent that every external influence calls it into activity, and causes motor impulses. M. Richelot declares in favor of the purely nervous theory. So much for the pathogeny.

The march of the disease is well described; M. Richelot gives first the general type of the affection, basing it on certain symptoms, the tonic convulsions, the variations of the temperature, the state of the pulse, the urine and the blood; then he passes in review the various admitted varieties, such as traumatic tetanus without wound, spontaneous or idiopathic tetanus, puerperal tetanus, tetanus nascentium, tetanus commencing at the wound, partial tetanus, and finally the slow and rapid forms.

In the chapter on terminations, the author considers in a few pages only the prognosis of recovery, of death, and the causes of the latter. He closes with a few words on the influence of treatment. "The surgeon should guard," says he, "at the same time against doing too little in mild cases, and in severe cases also. It is necessary, therefore, to treat tetanus actively. Thus far we have treated morbid symptoms and not the disease; but here the spasm has for its direct cause the disease itself and an agent that suppresses the one, proves by this that it touches the other."

The memoir closes with eleven hitherto unpublished observations showing the principal forms of tetanus met with in France.

SALTATORY REFLEX CONVULSIONS.—In 1859 Prof. Bamberger described (*Wiener Med. Wochenschr.*, No. 4 and 5), under the name of saltatory reflex cramp, two cases of a peculiar spinal irritation, and in 1867 Guttmann (*Berliner klin. Wochenschr.*, 1867, No. 13), described another. Dr. Anton Frey gives still another case at length in the last number of the *Archiv fuer Psychiatrie*, VI., I. p. 249, with remarks on the pathology of the affections at length. The phenomenon in question consists in a spasmodic lifting of the feet as soon as they touch the ground, this being repeated as long as the patient is held in an upright position.

The analysis of the symptoms of his case by Dr. Frey, and also of those previously reported by Bamberger and Guttmann, is given at considerable length. The symptom is seen in these, connected with quite different disordered conditions, but in all there was a general state of asthenia or a nervous predisposition. It could be suppressed by psychic influences and certain kinds of irritation. It seems to depend on a heightened reflex excitably caused by different morbid conditions, and from the fact that motility and sensibility were intact, and that the clonic convulsions are temporary, and during the intervals perfectly normal conditions existed. Dr. Frey thinks it quite certain that the columns of the cord are uninvolved, and that temporary functional disturbances existed only in the gray central substance and the commissures which have to do with reflex impulses. Summing up his conclusions, he says "the saltatory reflex convulsion is not a disease *sui generis*, but a possible form in very different spinal affections, depending on an increase of reflex irritability; and with all the variety of conditions in which it may occur, there is one point of simplicity: it appears, always when the patient attempts to stand upright."

LEAD PARALYSIS.—Dr. Ernst Remak, *Archiv fuer Psychiatrie und Nervenkrankheiten*, VI., Hft. I., in a paper on the pathogenesis of lead paralysis, maintains the theory that it depends upon lesions of spinal motor centres, a view favored by Erb in his work on peripheral nervous disorders, forming the first half of the XIIth volume of Ziemssens *Handbuch der Speciellen Pathologie*, and which in a modified form had been previously upheld by Tanquerel des Planches and Romberg. Dr. Remak gives a number of clinical cases in which the symptoms were very carefully analyzed, and the extent of the paralysis and the implication of various muscles very accurately tested, and finds that functionally related muscles are paralyzed without reference to their peripheral innervation. He also gives cases of spinal infantile paralysis in which the motor troubles were exactly limited to similiar functionally related muscular groups. While he admits that, especially in the lumbar enlargement, the localization of the spinal motor centres is very vague and defective, and needing further investigation, still in typical cases of lead paralysis of the upper extremity, he holds that there are sufficient data to enable us to seek the anatomical lesion in the anterior columns a little above the cervical enlargement, which clinical deduction as to the pathogenesis of the disorder will undoubtedly be supported by microscopic investigation.

The therapeutics of this disease should therefore include galvanization of the cervical cord, as has been recommended by Erb, together with that of the muscles and peripheral nerves.

ARTIFICIAL SOMNAMBULISM.—The following abstract by Prof. Math. Duval, of a recent memoir by M. Ch. Richet published in the *Journal de l' Anatomie et de la Physiologie* July 1875, is taken from the *Revue des Seiences Médicales.*

This interesting memoir is one of those that every physician and every physiologist or psychologist should read and meditate upon; it contains especially the accounts of experimental researches by the author, given, not as distinct observations, but as a successive study of various groups of well marked symptoms. What M. Richet designates as *somnambulism provoque* is nothing else than that vulgarly known under the name of animal magnetism, and more scientifically as hypnotism, Braidism, or artificial somnambulism.

In the first part of the article, the author gives a historical account of the psychic and somatic phenomena observed by him in artificial somnambulism; to produce this condition he does not have to have the attention fixed on some brilliant object, but he simply uses manual passes or strokes of the hand (which undoubtedly have the same effect) over the forehead and on the shoulders. Experience has shown that insuccess at first should not cause discouragement, and that in females we nearly always succeed if they consent to be tested the fourth or fifth time. First there is a torpor, followed by a true sleep with closure of the lids (under which the eyes are turned inward.) The somatic phenomena then observed are nearly *nul* or inconstant, while the psychic phenomena are of the highest interest.

"First of all I must state," says the author, "that I have never met with the pretended lucidity. I have tried, somewhat to my shame I confess, the improper questions to which the charlatans say the somnambulists make such precise responses, and have never yet obtained a satisfactory reply." What we observed is the condition of a sleeping subject who has consciousness of his state, and whose imagination, singularly excited, is let loose to all manner of dreams. All the organs of sense, with the exception of that of hearing, are dormant, and do not permit the external world to make any impression on the brain of the patient, but thanks to the persistence of hearing, the experimenter can make the somnambulist hear and understand him, and produce also such and such dreams which may induce successively or simultaneously hallucinations of sight, taste, smell or of hearing itself. Also, in spite of the great activity of the imagination and often the vivacity of the affective emotions, the subjects themselves are absolutely under the control of the will of those about them (will expressed in verbal orders.) They are true automata. The author lays much stress on this automatism, and the facts he reports in regard to this subject are not the least interesting of the memoir. His explanation seems to be the only true one, and it is based on the physiology of normal sleep. We need not give it here, but will only state that it is based on the fact that in sleep the will is absolutely subordinated to the imagination

In a second part of his memoir, the author examines the theory of simulation. He cannot find the absolute or pathognomonic proof of artificial somnambulism; but he accumulates evidences to show the absurdity of the hypothesis of constant simulation repeating itself always with the same phenomena. Having studied magnetic sleep as a disease, M. Richet has found it always the same, with a commencing period, a period of full development and a critical one, with symptoms both fundamental and constant, and accessory and inconstant. "Never having found," says he, "a subject resistant to its action, (I speak of women,) I cannot refuse to include it among the neuroses of the central nervous system.

The reader will find useful matter on the particular condition which psychologists have curiously studied in recent times, under the head of double consciousness (p. 362) We will conclude by giving here the conclusions that the author himself puts at the end of his memoir.

1. It is possible, by means of so-called magnetic passes, as well as by the fixation of the attention on a brilliant object, and by other little studied and inconstant empirical procedures, to produce a special neurosis analagous to somnambulism.

2. This neurosis, difficult to produce the first time, nevertheless is always brought about if we have the patience to attempt it in several consecutive seances. When once produced, it recurs again the more easily.

3. All the phenomena are in accord with teachings of physiology and psychology, and are found also in various degrees in certain intoxications and in certain neuroses of the central nervous system.

4. The truly characteristic phenomena are the hallucinations which can be produced whenever we desire, and a complete automatism, in such manner that the person is subject to the will of those about him, and experiences the imaginary sensations that they wish to communicate to him.

5. In presence of these constant facts, obtained under conditions always the same, we are forced to admit the existence of this neuropathy which differs from all others known, by its experimental production.

OCCIPITO-ATLOID TORTICOLLIS.—The following are the conclusions of a recent memoir by M. Dally, published in the *Bull. General de Therapeutique*, Nov. 15 and 30, 1875:

1. The articular deformities of the neck are due to affections primarily articular. They are not produced by contractures or functional spasms of the muscles.

2. Vicious positions of the head on the neck may be due to muscular action, but they do not produce permanent deformities nor even articular stiffness.

3. The hypothesis of active muscular contractions as a cause, is therefore deprived of foundation; the same, moreover, as in respect to club foot and scoliosis.

4. Muscular torticollis, as well as a certain number of cases of spasmodic torticollis, is cured by the methods of treatment directed especially to the muscles, electricity, localized gymnastics, communicated movements. etc.

5. The articular torticollis, causing muscular lesions, can be cured only by treatment addressed to the articulations,—that is to say that which re-establishes the normal relations of the articular surfaces, destroys the morbid adhesions and permits the muscles to take on again their normal action on the movements of the vertebrae; manipulations associated with the preceding methods.

6. There is a very characteristic kind of torticollis, originating from a displacement of the atlas on the occipital condyles. In this form, anchylosis seems to make its appearance from the first two months, and to resist treatment that succeeds in lesions or other segments of the vertebral column of equally long standing.

7. A very important matter in cases of acute torticollis, is to insure a normal attitude of the head on the atlas from the very first, to maintain this by all possible means, and to be prepared for the accident of an atloidean dislocation.

DIAGNOSIS OF THE POSITION OF THE LESION IN FACIAL PARALYSIS.—In a paper on facial palsy, of rheumatic origin in the *Deutscher Archiv fuer Klin. Medicin*, Dr. W. Erb gives some useful hints for determining the exact seat of the lesion of the nerve in any particular case which we re-produce here, for the benefit of our readers. His method depends on observing whether certain special branches of the facial nerve are affected or not simultaneously with those supplying the muscles of expression :—1. If the posterior auricular nerve is involved, the lesion is inside the canal of Fallopius. 2. If the sense of taste is diminished or destroyed on the corresponding side of the anterior portion of the tongue, the lesion is above the point where the chorda tympani leaves the facial nerve. 3. If the sense of hearing is abnormally acute for low notes and also for distinguishing small variations in musical tones, the nerve to the stapedius muscle, which is given off above the chorda tympani is implicated. 4. If the paralysis of the soft palate can be detected we may conclude that the lesion involves the trunk of the facial from the geniculate ganglion onward towards the brain. The application of these rules to a large number of cases has proved, according to Erb, that in the majority of instances of rheumatic facial paralysis, the seat of the lesion is lower than the point of origin of the chorda tympani, and that in the mildest forms, only that part of the nerve is affected which lies outside the canal of Fallopius; for being thus surrounded with loose tissue, it cannot be seriously compressed by the swelling of the neurilemma.—*Med. and Surg. Reporter.*

THE DISORDERS OF SPEECH IN GENERAL PARALYSIS.—The following are the conclusions of a recent paper by M. A. Voisin, published in the *Archives Gén. de Médicine.* We take them from the *Gaz. des Hopitaux*, No. 6:

En résumé, the troubles of speech in general paralysis consist in blundering, slowness, stuttering, hesitation, stammering, tremors and mutism.

The blundering, slowness, and the hesitation are due to cerebral lesions; the stuttering, stammering, and the tremor are the results of alterations in the medulla.

Mutism may be the consequence of cerebral lesions, and of lesions of the muscular and nervous apparatus of the tongue and lips.

The pathological physiology of these various disorders permits an anatomical diagnosis on the condition of the cases of general paralysis.

THE LOCALIZATION OF CEREBRAL DISEASE.—At the meeting of the Soc. de Biologie, Dec. 4, M. Joffroy offered an interesting communication bearing on cerebral localization, which, with the discussion that followed, is in substance thus reported in the *Gaz. des Hopitaux*, No. 148, 1875:

A man, aged thirty-four, presented every symptom of general paralysis of rapid development, which had come on within three or four months. In the latter part of this time especially the accidents succeeded each other with the greatest rapidity, the man presented a condition of great enfeeblement, constantly increasing; the sensibility being scarcely affected, while the disorder of the intellect was very marked. He had very great difficulty in speaking. A very marked tremor appeared in the muscles of the face, and the same phenomenon was observed in the arm of the left side. In the presence of such symptoms the diagnosis was easy, not dubious. During the last fifteen days the man had suffered from numerous apoplectic attacks, which were followed by a coma, persisting until death. On each buttock there was a scar, but that on the right was much longer than that on the left, and the two joined each other over the sacrum.

At the autopsy the meninges were found thickened, adherent in many places to the gray substance of the brain. Nevertheless it was rather easy to decorticate the anterior lobes of the two sides, but it was impossible for the posterior ones where the lesions were much more pronounced. These cerebral and meningeal lesions were especially marked in the posterior lobe of the left side. It was easy to see that they had made great progress in the later periods. M. Joffroy believed that there existed a manifest relation between the lesions of the posterior lobes and the lumbar eschars; in fact the cerebral lesion was most pronounced on the left side, and the scar on the right was more extensive than that on the left side.

In 1869, while *interne* under M. Charcot, M. Joffroy had observed an analogous fact; it was also in the case of a patient who died within a few days, after having undergone a general exhaustion with preservation of motility, and who was troubled with a deep and very extensive sore on the buttocks. At the autopsy there was found a very small hemorrhagic nucleus in the posterior part of the ventricle.

Recently at La Pitie he had again observed a similar case. A woman, aged forty, had an apoplectic attack, rendering her hemiplegic. This hemiplegia gradually disappeared, and movements returned, but at the same time the general condition became worse, and when brought to La Pitie her state was such that M. Joffroy thought at first that it was a case of typhoid fever; she was in a kind of stupor, talked with difficulty; there was a general weakness, and finally there was a single eschar on the left buttock. This patient died in a few days.

At the autopsy there was at first found no lesion of the brain, but by slicing it very thin into sections, a minute patch of softening, the size of a hazel nut, was found in the posterior portion of the right hemisphere.

M. Joffroy hence concluded from these facts that there exists a manifest relation between trophic disorders and the posterior portion of the brain; he deemed, also, that it might even be possible to determine what special region in the brain presides over trophic disorders. The lesions of the anterior portion are especially related to troubles of motility.

In concluding, M. Joffroy presented some considerations on the nature of the lesions that we observe in the cord in cases of trophic disorders, which consist in a secondary degeneration affecting especially the lateral columns.

M. Charcot observed that the questions of cerebral localization were still subjects for study. It is only in recent times that this question has been placed where it should be. In fact it is only in very recent times that we have found a process of study which permits us to arrive at truly scientific considerations. First of all, we must make ourselves thoroughly acquainted with the anatomy,—the topography of the brain. Up to the present time we have not been able to always define the locality of the lesion, but now in very many cases we can do this with accuracy. The parts of the brain that are least known are the posterior lobes; they are habitually silent as regards clinical manifestations. In cases of apoplexy for example, located in one of these lobes, what do we observe? A hemiplegia, mobile and transitory, frequently barely appreciable, and this is all, for there are cases unlike those mentioned by M. Joffroy in which we do not find eschars. Therefore in those cases in which we only observe this transient hemiplegia as a symptom, we have many chances of being right if we locate the lesion in the posterior columns.

M. Charcot declared himself a decided partisan of the theory of cerebral localization; he believed that he could safely affirm that certain lesions of the encephalon, perfectly limited and localized in certain points, always and necessarily produced the same symptoms. " But," he added, "we must also take account of lesions at a distance, and those affecting several points, at once; in these cases there may be variations, and they are not so clear. We have the diffusion through the whole reflex apparatus." M. Charcot applied to these facts all the ideas given out by M. Brown-Sequard, but there are cases in regard to which he did not share that opinion, and in which he believed there was a constant relation between such and such a lesion and such and such symptoms. For example, we cannot sever the cord without causing paraplegia.

Finally, M. Charcot considered that the subject of cerebral localization had been ill studied by the earlier observers; that it was a study to be taken up again, and one that had only commenced to be a little known very recently. Consequently we need give no heed to the older observations. They proved nothing to the eyes of M. Charcot, except that the older writers had observed poorly.

M. Brown-Sequard regretted that he was unable to agree with his friend M. Charcot. He admitted with him that a fixed lesion of a definite part of the brain gives rise to necessary symptoms. He was present to give a new proof of this fact by showing that puncture of the lumbar enlargement in the dog always gave rise to the phenomena of titubation of ataxia; in this point he agreed with M. Charcot. But the question is to know whether the destruction of this or that function corresponds with the irritation of any special part of the brain. M Charcot holds that a special point of the brain, the internal capsule, always produces paralysis. But there are cases on record of the destruction of the whole opto-striated body of one side without any paralytic phenomena.

M. Charcot should show that in these cases the facts were ill observed. The cerebral paralyses, according to M. Brown-Sequard, are phenomena similar to those which we observe when we galvanize the pneumogastrics in a dog; a cessation of activity consecutive to the irritation of a morbid portion of the brain.

M. Charcot stated that he regretted that he was not sufficiently acquainted with the dog, but he was pretty well acquainted with the human species in respect to the point under discussion. There are, he said, constant facts in the study of cerebral lesions. First he would throw out cerebral tumors as having no special importance relative to the phenomena under consideration. The only lesions that can serve to clear up this difficult study, are the more simple and circumscribed ones, such as the destruction of a part of the brain, the internal capsule, for example, by a hemorrhage; then we obtain direct phenomena, to use M. Brown-Sequard's own expressions.

But when we have to do with a tumor, a softening or a hemorrhage with effusion of blood into the ventricles, we meet with indirect phenomena. The case changes altogether; we have convulsions, complex accidents, and unexplainable symptoms. We then come under the rule laid down by M. Brown-Sequard. But this rule applies, according to M. Charcot, only in certain complex cases. Besides these there are others more simple, in which the localization of the lesions in certain central parts, and even in certain parts of the cortex of the brain, is perfectly demonstrable.

M. Brown-Sequard asked of M. Charcot what are the localizations which he allowed.

M. Charcot replied that as to that question he desired to remain as a pathologist; he did not care to enter upon the field of pathological physiology. He proceeded by empiricism, after a fashion, and not by experimentation, in the study of this important subject, and he believed he was safe in affirming in certain determinate cases, that such a lesion of the brain produced such a symptom if it was simple, and such a syndrome if it was complex.

M. Brown-Sequard then asked M. Charcot what was, in his view, the portion of the brain, the destruction of which produced the loss of movement.

M. Charcot answered that the destruction of the anterior two-thirds of the internal capsule inevitably caused loss of power of movement, and that of the posterior third caused hemi-anæsthesia.

In a following session of the Soc. de Biologie, Dec. 14, the discussion was resumed. M. Charcot, desiring to express himself more precisely than before, stated that a lesion of the anterior two-thirds of the internal capsule always caused hemiplegia, and that a lesion of the posterior two-thirds is accompanied with all the phenomena that he had described under the name of cerebral hemi-anæsthesia, *i. e.*, an alteration of all the senses of one side. Besides these localizations in the central masses of the brain, he admitted certain cortical localizations. Finally he agreed with M. Broca that the faculty of expressing ideas by words had its seat in the third frontal convolution.

M. Laborde called M. Charcot's attention to the fact that M. Veyssiere had observed in dogs a cerebral hemi-anæsthesia under experimentally produced conditions similar to those clinically observed in man, and that pathological experimentation which M. Charcot seemed perhaps to undervalue, thus came to his aid in support of his views. As regards the communication of M. Joffroy, M. Laborde stated that some cases of meningeal hemorrhage produced eschars which might serve to aid the diagnosis of the affected side of the brain, and hence they did not coincide alone with lesions of the posterior lobes.

In the meeting of December 18 the discussion was still continued.

M. Charcot first disclaimed any lack of appreciation of experimental physiology. He thought, however, that the opinions of physiologists as to the brain and spinal cord could not be accepted without reservations. The experiments on the lower animals had been of service, but it is from man, or his nearest ally, the ape, that the facts of value to the clinicist must be derived.

M. Brown-Sequard disagreed with M. Charcot, and to support his own views, he found himself obliged to detail to the society a number of observations. His design was to show that the cerebral localizations as held at the present are false. First, however, we must return to certain questions that govern the subject. The symptoms that we observe in cerebral affections, according to M. Brown-Sequard are the result of this or that lesion on the parts adjoining the injured region. Supposing for example a hemorrhage in the left third frontal convolution, we may observe two things, convulsions and aphasia, that is, an accident, the convulsions and the loss of a function, that of expression by speech. But in this case the convulsions are the result of a convulsivant power, located not in the seat of the lesion, itself, but in a region in its vicinity where the fibres passing from it end. The same mechanism will serve for the aphasia, and in consequence the third frontal convolution is not proved to be the centre for speech. For both facts, the convulsions and the aphasia, there is therefore the same mechanism and the same mode of production.

If we examine the mode of the production of the paralysis, said the speaker, we notice that it is, in the great majority of cases, the same as that of aphasia and convulsions. There are conductors in the bulb that serve for the transmission of the will to the muscles, if they are injured there will be loss of function, a loss of motility. This is true in most cases but the bulb may have other influences at a distance, which sometimes predominate and then the effects will be different.

If we study what takes place in the brain itself, the internal capsule in particular, we also see the importance of this influence at a distance in the production of paralysis. A great number of facts show that paralysis may occur without any lesion of these centres. On the other hand we see extremely limited lesions in a certain region followed by paralysis, and again we see very extensive injuries of the same part without it. Hence, we must conclude that these centres are different in different individuals, a conclusion that is absurd. There are cases in which a limited lesion of the brain causes a facial paralysis of the corresponding side, and others where it is on the opposite side. There are cases of alternate paralysis corresponding to lesions situated above the pons.

In the presence of these facts it is impossible to hold that the facial paralyses are independent alone on the injury of a motor centre for the face. In fact that would lead to the conclusion that this motor centre occupies all parts of the brain.

Finally, we have seen a limited lesion of the brain to produce, sometimes, paralysis of all the members on the same side, sometimes those of the opposite side, sometimes the upper and sometimes the lower members. M. Brown-Sequard did not insist on these facts, which, with the admission of the prevalent ideas, concerning localizations, led to absurd conclusions.

He would show further that, as concerns localizations, experimental observations supported entirely the pathological facts noticed in man. In fact the same variety of symptoms is met with in animals when the lesion is known and is produced under desired conditions.

M. Brown-Sequard commenced the enumeration of over two-hundred observations which tended to show the existence of hemiplegia with a lesion of the brain on the same side as the paralysis.

M. Charcot thought it impossible to continue this discussion under these conditions. Among the observations cited by M. Brown-Sequard there was not a single one that he did not find vitiated with all kinds of errors.

M. Brown-Sequard mentioned only exceptional cases, not those seen customarily, and attempted to build a theory upon them. For his own part, on the other hand, he would advance only well-known and usual cases, rejecting altogether the exceptional ones.

To speak only of the first case given by M. Brown-Sequard, M. Charcot said that the observation was disorder itself, and that it was impossible to draw any conclusion from it. The facts are contradictory and altogether exceptional and such as M. Charcot had never observed, in the sixteen years he had studied these questions, at the Saltpétriére. It would not be difficult, taking up each observation, to show that they were all defective, and to destroy all their value. The discussion is, therefore, impossible, since M. Charcot rejected all the facts on which M. Brown-Sequard supported himself. Adopting his colleagues own expressions, M. Charcot admitted that in the symptoms consecutive to cerebral lesions, it was necessary to distinguish direct, and indirect, phenomena. But the former are the rule and the latter the exception on which it is impossible to build general laws.

M. Brown-Sequard had intended to show, supporting himself on some hundreds of observations, the possibility of the production of paralysis of the side corresponding to the lesion. There was one well known, which M. Charcot could not deny, that of M. Diday (of Lyon.) Many others, as irrefutable. might be quoted. But a single exception is sufficient to render false a theory. Moreover, cauterization of the brain in animals caused the same result. He had communicated numerous observations in which cauterization of one side of the brain produced paralysis of the same side. We know that M. Longet explained these interesting facts by defect of the decussation in the pyramids, but this had never been met with by any anatomist.

M. Charcot repeated that in all the numerous cases he had had the opportunity to observe, he had never noted those contradictory to the theory he sustained. All those cited by M. Brown-Sequard, are therefore defective, either in a clinical or pathological anatomical point of view.

At the subsequent sessions of the society, the discussion was still continued. M. Luys, came to the support of M. Charcot against M. Brown-Sequard, stating that in all the autopsies he had made, he had never met with an exception to the rule that the phenomena met with during life always correspond to lesions of the opposite side. The cases on which M. Brown-Sequard supported himself, evidently did not meet the requisites of a valid case, as demanded to-day. He should, therefore, submit his contrary facts to the examination of his colleagues, and not be content with old observations of no value at the present time.

M. Luys then alluded to a class of facts supporting the views of M. Charcot,—the cerebral alterations consecutive to old amputations. He had found in autopsies of old persons, who had many years previously undergone amputation of the shoulder or a limb, atrophy of the opposite side of the brain, and had already collected a dozen of these cases. It was produced only some fifteen or twenty years after the amputation. Those parts of the brain which receive impressions from the member, and transmit to it the orders of the will are silenced, and like every disused organ, they undergo atrophy. He exhibited photographs illustrating some of these cases, which are additional supports of the law of the discussion.

M. Brown-Sequard said that it was exactly his intention to prove that cases of direct paralysis did really exist. This frequency had nothing to do with the question; a single instance would suffice, and he believed that he could quote an authority disputed by no one; he would cite a case published in the Archives of the Societe de Bologie, by M. Charcot himself. This was a case of tumor in the right hemisphere which during life produced a paralysis of the right side of the body, and he asked M. Charcot if he was not correct in his citation.

M. Charcot said that he recollected the case very well. The case was old, and M. Charcot of to-day, did not exclude M. Charcot of former times, from the number of authors whom he rejected relatively to the question of cerebral localizations. This observation, though his own, was of no more value than the others, except that it only dated back ten years. It should be remarked, moreover, that it was a case of tumor, and such were unfitted for this study, since they displaced the tissues without destroying them and were accompanied with no definite phenomena.

M. Brown-Sequard regretted to see M. Charcot so severe on himself. Experimentation on animals enabled us to decide this question incontestably: if we burn the surface of the brain on one side, we can discover that a certain number of muscles will be paralyzed on that side. But in these cases the lesion is produced voluntarily and we know that no others exist, or at least if there are others on the opposite side we know that they depend on the ones first produced, which are thus the primary cause of the paralysis.

M. Brown-Sequard next referred to cases in human pathology, such as aphasia with hemiplegia, in which we cannot do otherwise than connect the two symptoms, and related numerous cases of the kind. He stated, also, that there exist incontestible cases of cerebral hemorrhage in which the autopsy showed the lesion on the same side as the paralysis.

He presented before the society a brain of a dog which had undergone cauterization, that had been followed by direct paralysis. He had carefully examined it for other lesions but found none. He next begun to review the facts in favor of his view of the subject, reported by Cruveilhier, Bell, Diday, Desgranges, Hillairet, Gintrac, Rostan and others, and those of softening followed by direct paralysis, two cases of which were reported by Dechambre, and one by Henry Day. He also found support for his argument in the extent of the lesion; there are many cases in which nearly all of one hemisphere was destroyed, such have been reported by Messrs. Broca, Freschi, and Rostan. In other cases of wound of the brain, suppurating during life, paralysis of the same side existed, and others in which the paralysis diminished at once with the discharge of the matter, and it was impossible in these cases to refer the paralysis to anything but the abcess on the corresponding side. In another case a tumor of one side of the head caused temporary paralysis of the same side on the application of pressure.

M. Brown-Sequard then stated that he had already striven to show in numerous papers that a lesion of the base of the brain often causes paralysis of the corresponding side. He had more than fifty cases, in many of which the symptomatology was so clear and precise that he had been able to announce that the lesion would be found on the same side as the paralysis, and the autopsy confirmed this statement. There were also many cases in which a paralysis of the fifth nerve existed with a paralysis of the corresponding side of the body. In these cases the paralysis is usually incomplete; it occupies indifferently the upper or lower limb, is almost always accompanied by slight contracture, and often with special trophic disorders.

Besides these facts M. Brown-Sequard cited others apparently as decisive. In one case the pons was almost entirely destroyed and replaced by a tumor, with paralysis of the corresponding side; in another a tumor occupied a whole half of the medulla, with like result. A number of other cases might be cited.

After M. Feray had presented some facts relative to the topography of the brain, M. Charcot observed that none of the cases quoted by M. Brown-Sequard were observed with the anatomical precision possible with modern appliances and methods.

M. Pitre communicated three observations of cerebral lesions; the first was one of aphasia and right hemiplegia in which nothing was discovered at the autopsy until after the most minute and careful examination, when the lesion was affecting a portion of the third frontal convolution, the whole of which was found under the microscope to contain granular bodies. This case, but for the care in examining it might have been recorded as opposed to the theory of localizations. The other two cases were of lesions of the internal capsule diagnosed correctly *ante mortem*.

M. Charcot said these cases might be multiplied and he would return to them later. But in a general way we may to-day admit that there are certain symptoms which always correspond to lesions of the same part of the brain. Taking one part of the brain, the internal capsule, he divided it into two parts, one comprising its anterior two-thirds and the other its posterior third. If a destroying lesion occupies about the anterior third of the internal capsule we observe a motor hemiplegia, permanent, durable, and nearly always incurable. If on the other hand the lesion bears on the corpus striatum we have a transient motor hemiplegia. If it is in the posterior third the symptoms will principally consist in a permanent crossed hemi-anæsthesia, if the lesion is destructive; but if there is a hemorrhage, for example, in the thalamus or in the extra ventricular muscles, distending the surrounding tissues, we have then a transitory hemi-anæsthesia; in fact at a given time the internal capsule resumes its functions and the hemi-anæsthesia disappears. There are results that can be diagnosed without fail during life.

M. Brown-Sequard remarked that M. Charcot had himself published a case absolutely contradictory of his present views, a transient paralysis following a destructive lesion. It was published in the thesis of M. Lepine.

He then remarked that M. Vulpian had given a rational and possibly a correct explanation for many cases of direct paralysis, namely, the existence of a ventricular dropsy of the side opposite to the lesion, and had thus remitted these cases to the ordinary crossed paralysis.

But there are many cases in which the absence of ventricular dropsy has been determined, and others in which it was observed to be greatest in the ventricle of the injured side. M. Vulpian's explanation is therefore correct for some cases but not for others.

Another explanation has been offered of direct paralysis which has *a priori* apparently much value; it is the occasional absence of decussation of the pyramids. But we should first determine whether this decussation is as important as has been thought. M. Brown-Sequard had for a long time sought to show that this decussation included the fibres that carried the impulses of the will to the muscles. But he had since recognized that very few of these fibres are found in the pyramids at the level of the decussation. There are, moreover, many experimental and clinical facts that prove that the pyramids do not have so great an influence over voluntary movement: Magendie has shown that section of one pyramid has no effect on movement. The section of both causes a certain embarrassment to forward motion.

Schiff has made a complete section of the two pyramids, and found that voluntary movements were not lost nor even diminished. Vulpian made a longitudinal section of the medulla and found motility only a little diminished, not completely lost, as it should be if the pyramids were completely formed of motor fibres. M. Brown-Sequard himself had performed many experiments, and observed that section of one pyramid produced hardly any paralytic symptoms and that that of both was not followed by any very well marked paralysis. He concluded that they had little to do with voluntary movement, if anything.

As to the clinical facts he cited many cases in which lesion of the pyramids was without influence on voluntary movement. In one case of Vulpian's there was a lesion of the left crus and a notable atrophy of the anterior pyramid of the same side, without loss of voluntary movement; in another case all the fibres of one pyramid were destroyed without paralysis of the superior members (the lower ones being paralyzed on account of a myelitis co-existing with the cerebral alterations); two other cases were given one by Cruveilhier, the other by Bouchard. It results from these that the pyramids are not the sole routes of transmission of the orders of the will, and the default of their decussation is not enough to explain the existence of direct paralysis. M. Brown-Sequard here offered some new considerations on the origin of these direct paralyses.

He next called the attention of the society to the relation existing between convulsions and paralyses. According to him the production of a convulsion and that of a paralysis are two facts of the same order, dependent on the same cause.

He proposed to demonstrate in future communications that there is but one part of the brain, the absence or destruction of which is incompatible with the persistence of the cerebral functions. One-half of the brain alone sufficed for all these functions, in other words each half was functionally equal to the whole.

M. Charcot said that he agreed with M. Brown-Sequard in regard to the relation between contractures, paralysis, and convulsions. They are, so to speak, complementary phenomena. It is in the common cerebro-spinal meningitis that the relation of these phenomena to each other is especially to be observed. But he did not agree with M. Brown-Sequard in the interpretation of these phenomena as regards the lesions that produce them.

Being given, for example, a hemorrhage, when it occurs in the deep central masses, (the opto-striated body, the thalami, internal capsules) without touching the ependyma, there is never contracture, but if it affect the cortex then the first phenomena observed are contractures and convulsions followed by paralysis and resolution. These are the rule, based not on exceptional cases, but on facts of every day observation.

M. Laborde presented two dogs in whom he had produced cerebral hemorrhages under conditions as nearly similar to those in man as was possible. The first had been experimented upon a month ago, it still had a facial hemiplegia of the side opposite the lesion (hemorrhage near the left opto-striated body) a motor paralysis of the right side and a very well marked hemi-anæsthesia of the left. This dog had also almost directly after receiving the lesion a kind of very marked hemi-chorea of the right ear.

To-day these phenomena, still marked, are complicated with contractures of the two anterior members, especially noticeable in that of the left side, and rather pronounced ataxic symptoms of the posterior limbs.

The second dog, experimented upon in a like manner, presents only facial hemiplegia opposite the lesion and a hemi-anæsthesia of the body on the side corresponding to the injury.

The experiments were performed by drilling a minute hole in the skull and injecting blood by a small canula into the desired situation in the brain. The autopsies of the dogs would be reported at a future time.

M. Remond had likewise produced paralysis in dogs, but by another method.

M. Charcot said that when we speak of paralysis we should understand and distinguish the transitory from the permanent forms. When a hemiplegia, for example, is permanent, i. e, it lasts at least two months, and after that time motility begins to re-appear, we then see choreiform movements in the upper and lower members, exceptionally in the face; these movements are nearly always accompanied with hemi-anæsthesia, an exact reproduction of the hysterical hemi-anæsthesia. But this hemi-chorea called by M. Charcot, post hemiplegic, corresponds to a localization in the posterior third or fourth of the internal capsule, while the lesion which produced the hemiplegia occupies the anterior two-thirds or three-fourths.

M. Veyssiere, a pupil of M. Charcot, by injuring this same region directly in the dog, has been able to produce not only the hemi-anæsthesia but the hemi-chorea. Moreover, in studying the common form of chorea, M. Charcot found in hemilateral chorea, the frequent co-existence of a hemi-anæsthesia exactly comparable to that in hysteria. There exists, therefore, an evident relation between hemi-chorea and hemi-anæsthesia.

M. Brown-Sequard remarked the existence of a hemi-anæsthesia of the side corresponding to the lesion in the interesting observations of M. Laborde. In the case of superficial cauterization of the brain he had himself presented, he had obtained a diminution of the muscular sense on the same side, but not the complete disappearance of sensibility.

Continuing the discussion on Jan. 29, M. Brown-Sequard said that he had never met with a very clear case of paralysis without anaesthesia and even without hyperæsthesia. The study of the disorders of sensibility had been, according to him, too much neglected. Anæsthesia, alone, without paralysis, is rarely met with; out of eleven or twelve thousand cases he had met with only seven or eight.

In cases of direct paralysis, he had nearly always found also direct anæsthesia, and he cited numerous cases. There were, also, cases of crossed anæsthesia. He next mentioned some very rare cases in which only anæsthesia was observed, among others, one of M. Ollivier, in which a sudden loss of sensibility, lasting till death, without any motor trouble, was observed, and a case of Abercrombie, in which, also, the autopsy revealed a large abscess in the half of the brain corresponding to the lesion.

M. Brown-Sequard, in giving these facts, added, that he believed that he could say in regard to anæsthesias all that he had stated in regard to paralysis.

M. Charcot admitted that anæsthesia existed much more frequently than is generally believed in the affections of cerebral origin, permanent hemiplegias, for example. As in hysteria, it exists without the knowledge of the patient, and may pass altogether unperceived, unless sought for. Nevertheless, M. Charcot did not consider, with M. Brown-Sequard, the co-existence of anæsthesia with paralysis, as a rule; it would be easy to test the question at the Saltpêtriére, where there were never less than four hundred hemiplegics.

In the last reported discussion in this debate, M. Brown-Sequard called up the subject of convulsions in cerebral lesions, and stated that there were many cases of direct convulsions; *i. e.*, on the same side as the lesion. Lallemand explained these by the existence of a meningitis, and he himself had long favored this opinion, but had abandoned it when he found that convulsions depended upon other causes than affections of the cerebral membranes. In many cases of direct convulsions, the autopsy showed no trace of direct convulsions.

In reply to a question of M. Charcot, he said that authors did not speak always definitely in regard to the exact form of convulsions, some employing the term convulsions, others, that of convulsive movements, etc., but he cited only genuine cases.

He stated, also, that besides convulsions, there were also contractions, cataleptic attacks of prolonged spasm, followed, or more rarely preceded by clonic movements on the side corresponding to the lesion. Some observations, he said, were of incontestable value. When, for example, death follows rapidly a fracture of the cranium, we cannot do otherwise than admit the connection between the accident and the subsequent phenomena.

Unilateral convulsions of the side opposite the lesion are so common that they must be noticed. M. Brown-Sequard remarked that, in these cases the seat of the lesion was very variable. He did not know of any part of the brain that might not cause them on one side or the other, or, both. It is the same with the face. There are, he said, infinite varieties in the grouping of the muscles that are successively convulsed from cerebral lesion; thus there may be convulsions of the face and the two upper extremities, or the face and the two lower limbs, etc. He would say the same as he had said of convulsions, in regard to paralysis, contractions, tremor, etc. He would show cases where lesions above the protuberance had given rise to deviation of the head toward the well side, the reverse of the usual rule. He applied these views to all the losses of cerebral functions to aphasia and to amaurosis. He had seen cases of aphasia with right sided cerebral lesions.

Next, taking up his own peculiar physiological ideas, M. Brown-Sequard maintained that the relations between the muscles and the will centers were altogether different from what is generally supposed. All the so-called motor fibres, he said, passing or not from cells, are not necessary for the accomplishment of a voluntary movement. A single one may suffice, and the same, he held, is true of the centripetal sensory fibres! He thought he had shown that the destruction of a certain number of conductors in the cord or medulla did not implicate functions. He held that a very small number of fibres served to keep up the communication required by any function, and that the left half of the brain alone sufficed for all the cerebral functions. He added, however, that we must take account of a sort of education of the left hemisphere, which allowed it to control the function of speech. He thought, also, that the cells of the right half of the brain controlled more than the left the nutritive functions. But, instead of holding that the cells. endowed with special functions are grouped together, he held that they were disseminated over the brain, and quoted some experiments of Flourens in support of this view; in which he gradually sliced away nearly all the whole cerebrum before producing paralysis.

The conducting fibres, therefore, between the brain and cord are capable, under any irritation whatever, of producing alteration at a distance, causing paralysis, etc., and in this way only can we explain, according to M. Brown-Sequard, the various phenomena observed in man.

M. Onimus had repeated Flouren's experiments, and agreed with M. Brown-Sequard as to the difficulty of admitting voluntary motor centres.

M. Charcot said the differences between man and the lower animals were so great that it was difficult to establish comparisons. The brain had a much higher function and importance in the former than in the latter, and absolute comparisons were impossible.

M. Brown-Sequard agreed with M. Charcot, and said that he depended alone on the facts observed in man for the support of his theory.

M. Onimus closed the discussion with the citation of some observations of automatic movements in hemiplegic patients.

The Pathogeny of So-called Congenital Deaf-Mutism.—In a communication before the Acad. des Sciences, Dec. 20, (rep. in *Bull. Gén. de Thérap.*), M. Tripier showed that a fifth only of deaf mutes were so from birth, and that four-fifths on the other hand only became so at the age of two or three years.

"But," said Mr. Tripier, "from the examination of twenty cases in regard to which I have collected sufficient data, and of which the *résumé* and the discussion will be found in the tables appended to this note, it follows that the subjects in whom deafness begin suddenly at the age of two or three years, and who are improperly classed among the congenital cases, present disorders of locomotion sufficiently marked to clearly justify the comparison of the pathogenic conditions of deafness acquired in infancy and the spinal paralysis called by Duchenne the *atrophic fatty paralysis of infancy.*

Among others, we may mention the following, as recent papers on the Pathology and Pathological Anatomy of the Nervous System:

Behier, On General Paralysis of the Insane, *Gaz. des Hopitaux.* (cont. article); **Mauriac**, On Neuralgic Herpes of the Genitals, *Ibid*, (cont. art); **Cameron**, On a form of Insanity which may be termed Toxiphobia, *Dublin Jour. of Med. Science*, Feb.; **Balfour**, On Obstinate Trigeminal Neuralgia as a Symptom of Syphilitic Cerebral Disease, its Diagnosis and Treatment, *Edinburgh Med. Journ.*, Oct. 1875; **Schultze**, Secondary Degeneration of the Spinal Cord, *Centralblatt f. d. Med. Wissensch.* No. 10; **Goltdammer**, On Spinal Apoplexy, *Virchow's Archiv*, LXVI, 1, Jan. 26; **Elischer**, Alterations of the Brain and Spinal Cord in Tetanus, *Ibid;* **Schultze**, On the Condition of the Spinal Cord and the Spinal Nerve Roots, *Ibid;* **Bertelot**, Meningitis Tuberculosa in Children, *Jahrb. f. Kinderheilkunde*, IX. 3, Jan., 1876; **Neelsen**, On a Peculiar Degeneration of the Cerebral Capillaries, *Archiv der Heilkunde*, XVII, 2 and 3, Feb.; **Baumgarten**, A peculiar Case of Acute Ascending Paralysis, with Bacteria in the Blood, *Ibid;* **Kolesnikoff**, Alterations of the Nervous System in Hydrophobia, *Centralblatt f. d. Med. Wissensch.*, No. 50, Nov. 20th, 1875; **Schuele**, On Paralysis, *Allg. Zeitschr. f. Psychiatrie*, LXII, 6; **Mitchell**, Some Disorders of Sleep, *Va. Med. Monthly*, Feb.; **Wood**, Two Clinical Lectures on Cerebral Syphilis, *Phil. Med. Times;* **Mitchell**, Notes on Headache; Headaches of Anaemia; Headaches of the Decline of Life, *Med. and Surg. Reporter*, Feb. 5.